Figure 1. Morphs of the freshwater snail *Melanoides tuberculata* that have invaded Martinique.
Average fecundity (as the number of individuals produced during one week) and size of two invasive morphs (FAL and PAP) as well as their sexually produced offspring (CPF). The sexually produced morphs are significantly bigger and may also have a fitness advantage based on increased genetic diversity stemming form multiple invasions. (Photographs kindly provided by Laurent Soldati and Benoit Facon.)

Furthermore, the new work demonstrates a shift in the life-history strategy within the two new sexually produced morphs: they have lower fecundity but their offspring are larger than that of the parental morphs. For example, one cross between two introduced morphs yielded a morph with a mean fecundity of less than half that of its parents, while the mean size of the offspring has more than doubled (Figure 1). The sexually produced morphs have replaced their progenitors in several streams of Martinique, suggesting that this new life-history strategy provides higher total fitness in this environment [14]. Thus, the multiple introductions of distinct morphs provided a dramatic range of phenotypic variation, while outcrossing created entirely new, transgressive phenotypes, perhaps through polyploidization.

This new research [8] supplies, for the first time, evidence that transgressive variation created via hybridization can facilitate invasion. It further strengthens growing evidence that multiple introductions and genetic admixture can have real evolutionary consequences for invasive species [7,15], with the potential to radically alter the expression of key life-history traits and provide variation for further fine-tuning to a new environment [16]. Biological invasions can no longer be seen as genetic paradoxes. Rather, they are crucibles for evolution.

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Cytokinesis: Catch and Drag

Recent studies of actomyosin-ring assembly in fission yeast have suggested that an intricate web of membrane-bound nodes containing myosin and the actin nucleator formin is pulled together into a tight ring through a 'search-and-capture' mechanism.

Mithilesh Mishra and Snezhana Oliferenko*

Actomyosin-based 'rings' underlying the plasma membrane are assembled between segregating copies of genetic material during eukaryotic cell division. Sliding of actin filaments by myosin motors leads to ring constriction, which in turn is thought to permit cortical ingression and individuation of daughter cells. The actomyosin rings are dynamic, complex structures and their assembly and constriction in the plane orthogonal to mitotic spindles are

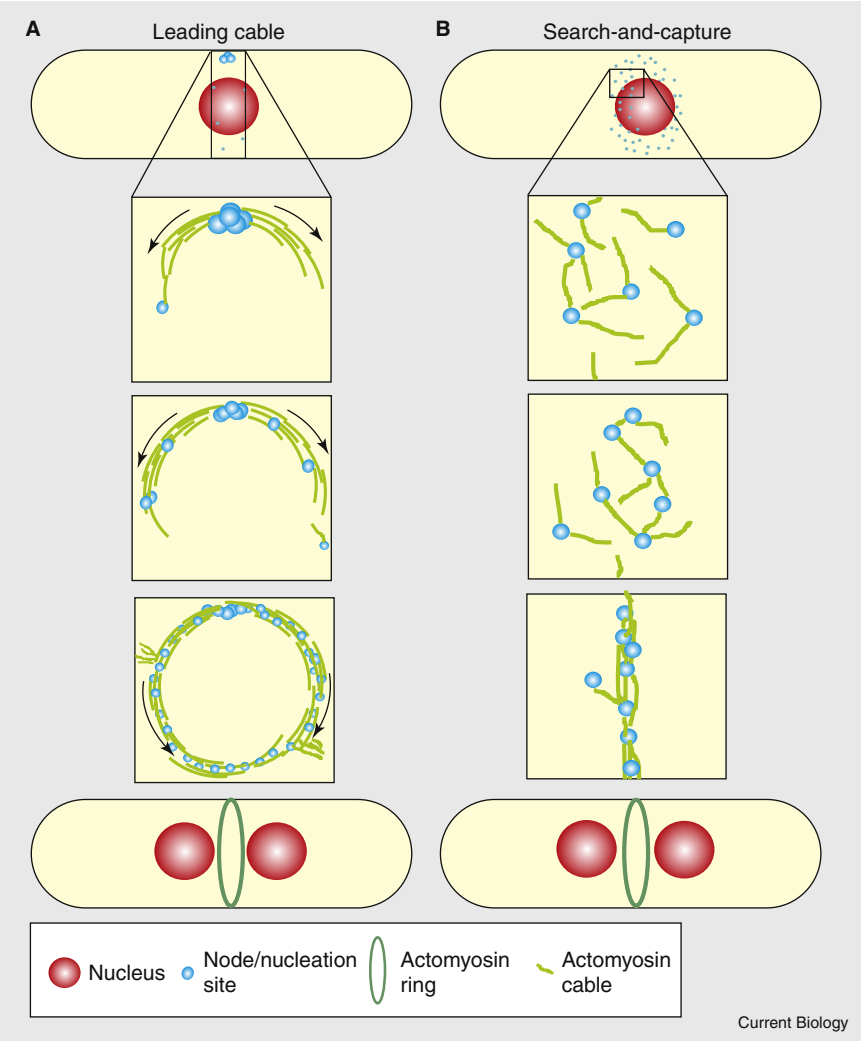

Figure 1. Models of actomyosin ring assembly.

(A) The leading cable mode of ring assembly invokes ring assembly from actomyosin cables primarily emanating from a single nucleation site. (B) The search-and-capture mechanism suggests that a broad network of cortical 'nodes' containing myosin and formin coalesces into a focused ring underlying the plasma membrane of the fission yeast cell, through tension generated by myosin molecules on adjacent nodes.

tightly regulated in space and time. Each cell cycle, the division rings are assembled *de novo* with remarkable efficacy and speed. So the question arises: how are rings made?

The fission yeast *Schizosaccharomyces pombe*, like metazoans, sets the site of future division and assembles division rings consisting of myosin, actin and associated proteins upon entry into mitosis. Studies in *S. pombe* have defined an evolutionarily conserved set of proteins important for actomyosin ring assembly, determined the genetic dependencies of their localization and function and created a wealth of cell biological and biochemical data pertaining to the behavior of individual ring components [1,2]. Thus, the time is ripe to attempt a quantitative, systems-level understanding of ring assembly and function.

Current ideas regarding mechanistic underpinnings of the ring assembly can be grouped into two classes, although some proposals incorporate features of each mechanism [3]. According to the 'leading cable' hypothesis, division rings are primarily formed from actomyosin cables emanating from a single point at the cell cortex and curving around the cellular circumference (Figure 1A). One could imagine that bidirectional cables slide along the medial cortex, which is defined by the anillin-like protein Mid1p, and get cross-linked or pulled together into a compact ring overlying the nucleus [4–7]. An alternative model first proposed by Bahler *et al.* [8] and developed by Pollard's group [9] postulates that Mid1p organizes a number of nodes (typically ~60) at the equatorial cortex that further recruit an actin nucleator (the Cdc12p formin), type II myosin, the IQGAP protein Rng2p and the EFC domain protein Cdc15p. It has been proposed that each node could both nucleate actin and capture actin filaments emanating from other nodes. Force generated by myosin would then be used to coalesce the broad medial network into a tight ring structure [9] (Figure 1B). This latter hypothesis has now received support from further studies from Pollard and colleagues [10] that bring together impressive *in vivo* imaging of ring compaction and mathematical modeling of this process.

By analyzing the behavior of both actin and myosin constituents with fine spatiotemporal resolution, Vavylonis *et al.* [10] could detect previously unappreciated events in the ring dynamics, such as the nucleation, growth and severing of individual actin filaments in relation to myosin-containing nodes. It appears that each node can, on average, nucleate two actin filaments at any given time and that these filaments can extend in any direction. When filaments are captured by adjacent nodes, they can move towards each other, presumably through the motor function of myosin II. The transient nature of this interaction results in short-range movements. On the basis of these observations, the authors built a physical model of compaction of the initial network of myosin nodes that are connected by linear actin elements. Numerical simulations testing this model suggest that this random 'search-and-capture' mechanism could indeed lead to efficient ring compaction within the confines of *S. pombe* cell geometry.

This proposal is certainly an attractive idea that allows robust self-organization of a complex macromolecular structure. Intuitively, by broadly distributing the network elements and making them highly dynamic, cells could ensure rapid error-free ring assembly. The stochastic nature of interactions between nodes and linear elements endows the process of ring formation

with more plasticity and efficiency than would be expected from nucleating the ring from a single point.

There are two important considerations that will need to be addressed in further refinements of the search-and-capture model. First, the model predicts that a substantial fraction of actin filaments in the assembling ring is arranged in a non-uniform manner. It is indeed difficult to determine the orientation of actin filaments constituting the ring using fluorescence techniques. However, Mabuchi and colleagues [7] have recently reported the arrangement of actin filaments during ring assembly and constriction at the ultra-structural level. Stunning three-dimensional reconstructions of early anaphase rings, with the actin filaments decorated by myosin S1 fragments, revealed that rings are composed of two semicircles of predominantly parallel actin filaments of reverse orientation. This observation has been interpreted as consistent with the leading cable model, where the initial ring structure is nucleated in a bidirectional manner from a single site at the cortex.

Nonetheless, while the search-and-capture mechanism aims to explain the initial stages of ring formation, the only available ultrastructural data describe the already compacted rings. It is possible that single actin filaments connecting adjacent nodes are either absent from rings that have coalesced or they cannot be imaged by electron microscopy with sufficient contrast. If so, the micrographs by Mabuchi and colleagues [7] may show the larger actin bundles that contribute to stabilization rather than actual compaction of the actomyosin ring. The search-and-capture hypothesis hinges upon the contractile activity of myosin during ring compaction. Fittingly, the UCS domain protein, Rng3p, is required for both myosin gliding motility *in vitro* [11] and incorporation of cortical myosin spots into a ring structure [12]. Rng3p is not detected in coalescing rings despite being present at a higher cellular concentration than, for instance, Cdc12p and Mid1p [2], but it is possible that Rng3p–myosin interactions are transient and highly dynamic. Thus, it would be of interest to pinpoint the exact cellular defects during early stages of ring assembly in cells lacking Rng3p function: do cortical nodes nucleate actin filaments? Are these filaments captured? Also, how would nodes coalesce in the presence of defective myosins that have compromised motor activity? For instance, removal of IQ domains in the neck region of myosin heavy chain leads to severe defects in actin gliding *in vitro* [11], but cells expressing such mutants as the sole source of myosin can still assemble division rings [13]. Again, it might be interesting to describe fine actomyosin dynamics in such a situation and to analyze potential outcomes predicted by the search-and-capture model.

Another important prediction of the search-and-capture model is that the number of the nodes and the spacing between them is critical for ring assembly. Notably, fission yeast cells lacking Mid1p do not assemble detectable nodes [9,14]. However, the mutants are viable and form actomyosin rings [8,15,16]. Ring assembly in *mid1*Δ cells is somewhat delayed and rings are initiated as several actomyosin cables that eventually compact into a ring [14]. Thus, rings can be formed without the cortical network of microscopically visible nodes, possibly through the mechanism proposed by the leading cable model. Presumably, both types of ring assembly would have distinct molecular and temporal requirements, again providing fertile ground for future investigations.

Models describing complex networks could be susceptible to hidden variables and functions of components that are as yet unidentified. For instance, the search-and-capture mechanism assumes that tension generated by the myosin motor on actin filaments switches off the formin-driven actin polymerization [10], but this assumption differs from previous proposals [17]. Reconstitution of the myosin drag on actin filaments polymerizing from immobilized formin molecules could be a direct test for this assumption. All in all, it would be important to continue refining the search-and-capture model as new mechanistic studies of other ring components become available. Furthermore, a plethora of cytokinesis mutants generated in *S. pombe* should allow for its rapid testing.

The search-and-capture mechanism of division ring assembly provides an exciting entry into the busy field of cytokinesis studies that are being carried out in fission yeast. It remains to be seen whether a similar mechanism could operate during assembly of the division furrow in metazoans or whether it could be possibly aided by additional factors that function in the context of larger cellular volumes. Importantly, high resolution imaging techniques and physical models developed by Pollard and co-workers provide a framework for further analyses of mechanisms of ring assembly and function.

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Colour Vision: Cortical Circuitry for Appearance

Directly stimulating certain cortical neurons can produce a color sensation; a case is reported in which the color perceived by stimulation is the same as the color that most effectively excites the cortical circuitry.

Brian Wandell

One of the great achievements of neuroscience is the complete description of the early stages of color vision. In the human retina there are three types of cone containing different light absorbing pigments, each with its own unique sensitivity to the wavelengths of light. Because we acquire only three cone-type samples, and thus critically under-sample the available wavelength information, we are quite poor at resolving the wavelength information in a scene. The design of nearly every modern imaging technology — from displays to printers to cameras — takes advantage of the fact that humans encode light using only three types of cone. Technology standards show us how to capture and display enough information to persuade the cones that they are looking at the original scene [1].

Cones are clearly central to color vision, but the relationship between cone responses and our color perception is not straightforward. Retinal and cortical circuits process the cone responses to create our experience of color. These processes can be revealed by visual demonstrations in which the same cone photon absorptions produce different lightness and color appearance (Figure 1). Some principles of the neural coding — most importantly, the fact that the cone signals are recombined in the retina into three channels, known as opponent-colors, which are made up of sums and differences of local cone responses — are also used in engineering standards, including television transmission and image compression.

But, we do not have theories that accurately predict the patterns of color we perceive. How cortical circuitry interprets the encoded information remains a grand challenge for color science. For many years, the location of the essential cortical circuitry of color was a very contentious point, with many investigators doubting the very existence of any cortical specializations for color. Neuroimaging and neurological case studies over the last century demonstrate that signals in ventral occipital cortex (Figure 2) are essential for the perception of color [2]. For example, responses in a portion of ventral occipital cortex rise and fall as subjects alternately view colored and luminance-matched achromatic objects [3]. Damage to these same regions of cortex produces a syndrome known as cerebral achromatopsia — a color disturbance of cortical origin. Rather remarkably, in this syndrome color perception is severely altered without any obvious interference with other abilities, such as form, motion or depth perception [4,5].

In a paper published recently in *Current* Biology, Murphey *et al.* [6] provide a glimpse into the relationship between brain activity, brain stimulation and color perception. Their work bypasses the intricate color machinery of the retina and cortex. Instead, they study a patient who had an electrode array implanted in order

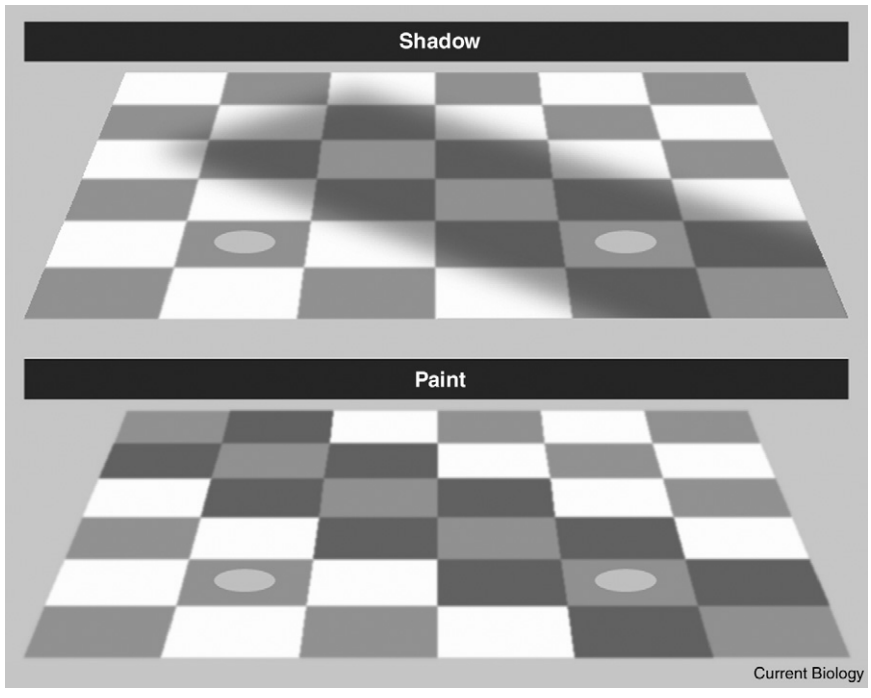

Figure 1. Two images which are identical apart from the shadow penumbra.
In the second image, the penumbra is replaced by a sharp edge coinciding with the checkers. Most people see a greater difference in the lightness of the spots in the shadow (top) than in the paint (below). The appearance difference is not caused by differences in cone signals, but rather by the neural circuitry's analysis of the absorptions. (Reprinted from [16].)